

Parameters measured and calculated within the fetal cardiac cycle

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Many attempts have been made to associate particular FHR patterns with certain types of fetal stress, though it is common clinical knowledge that head compression, umbilical cord clamping and hypoxia can give rise to similar FHR patterns.

Since there is no means of associating different fetal stress situations with typical FHR patterns, the application of more recent computer methods and techniques seems to be a promising area in which to achieve further information on the physiological condition of the fetus.

The measurement of the fetal systolic time interval (STI) shows a relationship with myocardial contractility. The knowledge of changes in myocardial contractility may supplement the interpretation of FHR patterns as an indicator of the presence or absence of fetal distress.

The time period from the onset of ventricular depolarization (QRS complex or deflection of the R-wave from the isoelectric line) to the onset of ejection from the left ventricle is known as PEP. Since PEP consists of two parts and the interval Q-Mc, where Mc stands for the closure of the atrioventricular (mitral) valve, is fairly constant in the fetal cardiac cycle, PEP will vary with the isovolumetric contraction time (IVC). As myocardial contractility (dp/dt)_{max} increases, PEP will decrease. The total interval PEP is inversely proportional to the rate of rise of ventricular pressure. In other words, PEP is an index of left ventricular function and reflects changes in myocardial contractility, left ventricular enddiastolic volume and aortic diastolic pressure. Therefore the continuous measurement of STI, especially PEP, may provide the first direct clinical assessment of fetal myocardial function. Several investigators including ourselves have found that continuous changes in STI should enable discrimination between different fetal stress situations, especially those due to uteroplacental insufficiency.

We have studied STI in 22 mature lamb fetuses exteriorized by caesarean section with the umbilical circulation intact. The results concerning the PEP during these acute animal experiments are summarized in Fig. 4. The changes of PEP and FHR in nearly all trials on head compressions were inversely. It seems that PEP is prolonged in proportion to the applied pressure. In nearly all of our experiments concerning umbilical cord clamping the PEP became prolonged too, but depending on the fetus condition, it shortened well below control upon release. These changes were associated with a decrease in FHR and with a marked rise in the mean arterial blood pressure. On the other hand in all our experiments, when the ewe was ventilated with nitrogen in order to produce fetal hypoxemia the PEP shortened. The relation between duration of ventricular ejection time (VET) and R-R intervals from 8 fetal sheep are shown in Fig. 1. VET is the interval between semilunar valve opening and closure. VET increases as R-R increases from 225 to 525 msec. (Heart rate decreases from 267 to 114 bpm). No further prolongation of VET was observed with R-R intervals greater than 525 msec Mean + SD.

The VET chiefly reflects the isotonic phase of cardiac work. This time interval is closely related to heart rate, stroke volume and, indirectly, to afterload and contractility. In order to come to more detailed understanding of our results, we estimated the stroke volume (SV), heart minute volume (HMV) and the total peripheral resistance (TPR) from the arterial pressure wave [1]. Fig. 2 shows a typical trial of hypoxemia on one fetus by replacing for 3 min. the maternal oxygen supply with 100% nitro-

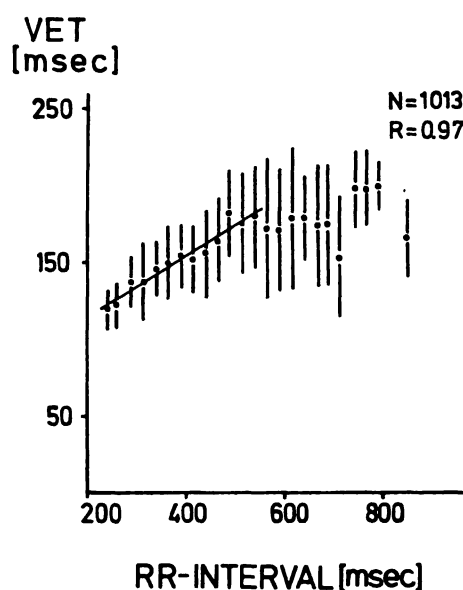
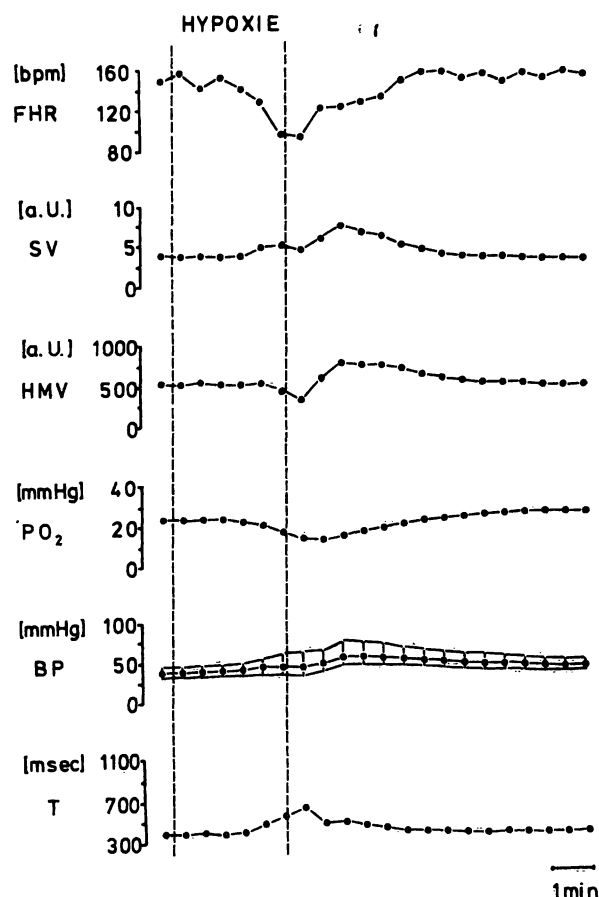


Fig. 1: Relation between duration of VET intervals and R-R intervals from 8 fetal sheep.

Fig. 2: The course of a trail of hypoxemia is shown. The pO_2 was measured continuously in the aorta. T is the period of the heart cycle.



gen. In association with FHR deceleration the fetal arterial blood pressure rose, especially the systolic (S) and PEP (not depicted) and the arterial pO_2 fell. In some of these trials our calculations showed a constant TPR and the SV increased. In so far as our methods to evaluate TPR, SV and HMV are reliable, the hypertension in SV as demonstrated in Fig. 3 is not in agreement with the findings of RUDOLPH and HEYMANN [2]. With respect to the FHR levels of 120 and 180 bpm we found the sequence of decreasing, increasing, decreasing SV values with increasing FHR. These results from 3 animals are calculated from 7 trials of fetal head compression. In Fig. 4 the results are summarized. The tip of each triangle shows the direction of change caused by the event. Triangles marked out are reproduced in nearly all of the experiments. The two triangles in the column blood pressure (BP) demonstrate the superelevation of the systolic (S) as seen in Fig. 1. SV and especially PEP seemed to be sensitive parameters when the O_2 -supply was lowered.

Our findings in earlier experiments [1] that the time from the QRS-complex to the first heart sound (S1) and PEP is correlated ($R=0.89$) led us to the conclusion that it is satisfactory to measure Q-S1 instead of PEP. And this non-invasive method offers a great clinical advantage in the study of hemodynamic changes of the fetal cardiovascular system.

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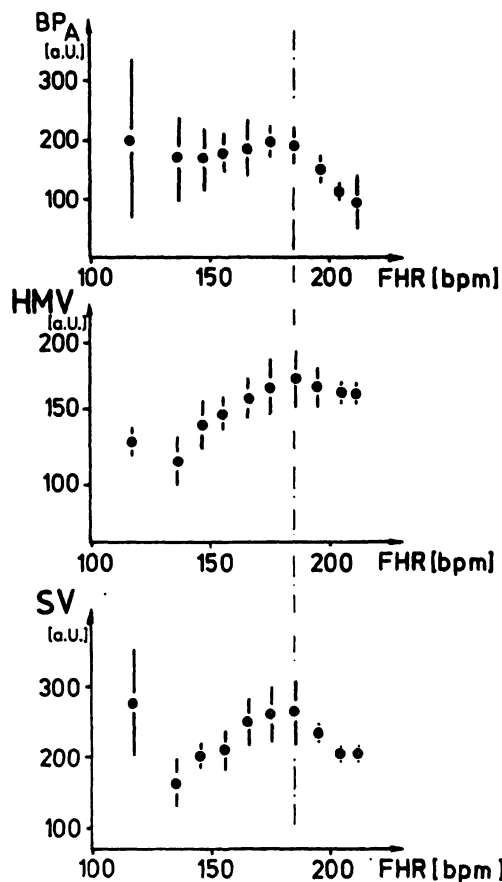


Fig. 3: Relationship between blood pressure amplitude (BP_A), heart minute volume (HMV) and the fetal heart rate (FHR), respectively.

Fig. 4: Schematic changes of the parameters due to different events. In the column blood pressure (BP); S, D stand for systolic and diastolic, respectively.

	PEP	SV	CO	BP S D	TPR
HEAD - Comp.	▲	▼	▽	▲▲	▲
UMBILICAL CORD Clamp.	▲▼	▼	▽	▲▲	▲
HYPOXIE VENA CAVA Comp. mat. N ₂	▼▼	▲	△△ △△	▲▲	△△ △△

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